



Wound haematoma: The first sign in a case of late postpartum HELLP syndrome☆☆☆



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ABSTRACT

HELLP syndrome, a severe manifestation of preeclampsia characterised by haemolysis, elevated liver enzymes, and thrombocytopenia, occurs in 0.5–0.9% of pregnancies and is associated with significant maternal and fetal morbidity and mortality. We present the case of a 30 year old primigravida (RL) who developed a wound haematoma nearly 72 h after an emergency caesarean section for failure to progress, with no prior hypertension or proteinuria documented. Although RL remained completely asymptomatic, investigations for delayed bleeding revealed severe class I HELLP syndrome with a platelet count of <50,000 μL , significant haemolysis (haptoglobin <0.06, LDH 1585), acute renal failure (eGFR 64, creatinine 103), fulminant hepatic failure (AST 2539, ALT 3200) and significant autoanticoagulation (INR 3.2, activated prothrombin time 46, fibrinogen 3.0). Paracetamol had been administered for post-operative analgesia and a paracetamol level was in the toxic level. Multidisciplinary input was sought from anaesthetics, intensive care, toxicology, general medicine, haematology and gastroenterology, with care subsequently coordinated in an intensive care unit. Blood pressure was strictly controlled with a sodium nitroprusside infusion. In addition to supportive care, vitamin K, a N-acetyl cysteine infusion, lactulose and mechanical thromboprophylaxis were administered. Eight weeks postpartum there were no residual biochemical abnormalities, the patient was well, and had a normal blood pressure. Our case reinforces the importance of a high level of clinical suspicion for the HELLP syndrome in women, irrespective of blood pressure in the first 48 h postpartum.

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1. Introduction

Preeclampsia is a condition unique to human pregnancy underpinned by either fundamentally deficient placentation or placental dysfunction. This, via multiple pathways, leads to multiple organ system manifestations of widespread endotheliolysis/endothelial dysfunction, stereotypically identified with, but not limited to, high blood pressure (hypertension) and protein in the urine (proteinuria). 0.5–0.9% of pregnant women will develop HELLP syndrome, a severe manifestation of preeclampsia characterised by haemolysis, elevated liver enzymes, and thrombocytopenia [1].

Although risk factors for preeclampsia (including later maternal age, body mass index greater than 35, smoking, family history, multiple gestation and primiparity) have been identified, and the field of preeclampsia screening is an exciting and evolving area, there are as yet

no routinely administered effective methods of detecting those who are at risk of developing this serious condition [2]. As approximately 30% of women who develop HELLP syndrome will develop it postpartum, a high level of clinical suspicion is paramount [3,4].

2. Case report

We present the case of a 30 year old gravida two para one (RL) who developed a wound haematoma 72 h after an emergency caesarean section for an obstructed term spontaneous labour. RL had a history of one prior first trimester spontaneous miscarriage and no relevant medical or surgical history. Antenatal history was complicated by bothersome hyperemesis gravidarum in the first and second trimesters requiring short admissions for intravenous rehydration, and a low pre-pregnancy BMI with poor gestational weight gain but was otherwise entirely unremarkable. The patient had a booking blood pressure of 110/60, and was normotensive throughout the pregnancy. Booking and 28 week bloods were unremarkable. A pre-caesarean full blood count was haemolysed and not repeated as RL was normotensive and thrombocytopenia not clinically suspected.

RL presented in spontaneous labour at 40-weeks and 5-days gestation, and delivered a healthy 3.1 kg male by caesarean section after

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a labour obstructed at 6 cm. The procedure was uncomplicated, with minimal blood loss under spinal anaesthesia. The initial postpartum period was unremarkable, with blood pressures persistently below 120/80 for the first 48 h. On day three postpartum, an isolated blood pressure of 132/90 was recorded, taken with a standard sized adult cuff on a small adult arm. Later that day medical review was requested for a wound mass noticed by the patient, and a diagnosis of rapidly expanding wound haematoma was made. A full blood count was requested as the initial investigation and identified a platelet count of 52. A full pre-eclampsia screen followed, including a repeat confirmatory full blood examination and film, serum uric acid level, liver function testing, measurement of serum creatinine, urea and electrolytes, urine for spot protein creatinine ratio, and a coagulation profile (Table 1).

No symptoms other than haematoma were reported by the patient, and other than the isolated borderline blood pressure, no clinical signs were present to reflect the eventual diagnosis with no headache, epigastric pain, visual changes, neurological irritability, hyperreflexia, clonus or oedema.

Urgent investigations revealed multiple significant biochemical derangements consistent with a diagnosis of HELLP, with acute hepatic failure (AST 2539, ALP 3200), acute renal failure (eGFR 64, creatinine 103), a positive haemolytic screen (LDH 1585, haptoglobin <0.06), and significant coagulopathy (INR 3.2, APTT 46, fibrinogen 3.0).

RL was admitted to the intensive care unit for ongoing investigation and management. Simultaneously, blood pressure began to rise rapidly up to 160/100, and was controlled with a sodium nitroprusside infusion.

As is customary for the post-caesarean state, RL had been taking regular paracetamol for analgesia since delivery and with undiagnosed impaired liver function required active management of a toxic paracetamol of 347 µmol/L, requiring an infusion of N-acetyl cysteine. This supratherapeutic level occurred through oral administration of paracetamol at recommended dosages, with no intravenous administration. Other care was primarily supportive. Lactulose was given for liver failure and hepatotoxic medications were withheld. Vitamin K was administered to reverse her coagulopathy.

Investigations by medical, intensive care, haematology and gastroenterology teams revealed no other cause for RL's presentation.

An abdominal ultrasound showed a normal liver, no intra-abdominal pathology, and a haematoma under the caesarean wound above the muscle layer. RL was managed by a multidisciplinary team including gastroenterology, haematology, obstetrics, toxicology and

intensive care. The worst biochemical state was reached approximately 75 h postpartum, and the patient steadily improved over the next week.

The patient was discharged from the intensive care unit after four days, with dramatically improved liver function, renal function and coagulation profile (Table 1). RL was followed up six weeks postpartum with a consultant obstetrician and had no residual issues, and completely normalised blood and urine results. Blood pressure was entirely normal without medication and she was asymptomatic and well.

3. Discussion

Although HELLP syndrome can occur postpartum, and is not preceded by hypertension or proteinuria in up to 10–15% of cases [5], our case was unusual in that the patient felt well and had no preceding signs or symptoms. Review articles suggest that up to 90% of cases present with epigastric pain as an early symptom, 90% with malaise, and 50% with nausea or vomiting [6]. In our case, despite significant biochemical abnormalities, the patient was completely asymptomatic, and was investigated only for a wound haematoma on what would have customarily been the day of discharge.

Identification and presentation would likely have been further delayed with a vaginal delivery as hospital discharge occurs 1 to 2 days earlier and would have only represented when grossly symptomatic. It is rare to have an asymptomatic, late postpartum case of HELLP syndrome with a woman who had been normotensive without proteinuria throughout the pregnancy, and reinforces the importance of a high level of clinical suspicion to ensure that a timely diagnosis is made. Additionally, this case demonstrates a salutary lesson of ensuring appropriate sized blood pressure cuffs available in a ward setting.

Class I HELLP syndrome, or those with haemolysis, liver dysfunction and thrombocytopenia with a platelet count at the nadir of less than 50,000 µL are associated with the highest levels of maternal morbidity [7]. However, it is important to be proactive and monitor carefully all women with HELLP syndrome regardless of their pathology results, as not all laboratory parameters are independent risk factors for adverse maternal outcomes [8]. Although we were fortunate in our case that no lasting effects occurred, a case report of postpartum HELLP syndrome led to a maternal death, despite timely intervention, with a similar biochemical profile to our patient [9].

The paracetamol toxicity that occurred in our patient was likely to be a result, rather than cause, of the liver dysfunction, as it had been administered at recommended dosages. However, this again highlights

Table 1
Serial pathology reports. a, b, c, d, e, f

Hours after delivery	28/40	72	75	84	96	108	132	180	6 weeks
Haemaglobin	113	121	151	103	113	93	96	84	131
White cell count	10.1	13.4	9.8	13.7	12.1	8.9	12.1	10.8	5.2
Platelets	205	52	40	75	105	96	125	201	221
Haematocrit	.033	0.35	0.44	0.30	0.33	0.27	0.28	0.24	0.38
Urea			5.3	4.9	5.1	4.2	3.5	2.9	3.7
Creatinine			103	80	74	60	61	60	55
eGFR			64	86	>90	>90	>90	>90	>90
ALP ^a			325	261	307	240	253	311	95
GGT ^b			83	69	93	79	132	494	33
AST ^c			2539						
ALT ^d			3200	2588	2485	1840	1195	692	23
Bilirubin			101	77	101	72	96	96	17
Albumin			24	20	23	18	18	21	38
INR ^e			3.2			2.0	1.4	1.1	
APTT ^f			46			36	29	28	
Fibrinogen			3.0			2.7	3.1	3.6	

^a Alkaline phosphate.

^b Gamma-glutamyl transferase.

^c Aspartate aminotransferase.

^d Alanine aminotransferase.

^e International normalised ratio.

^f Activated prothrombin time.

the importance of a high clinical suspicion for HELLP syndrome and early diagnosis, as paracetamol administration had continued for longer without realisation, long term or irreversible liver damage may have occurred.

Approximately 8% of women with HELLP syndrome will also develop renal failure, as did our case. Development of HELLP syndrome postpartum appears to increase the incidence of renal dysfunction [3]. Development of acute renal failure is associated with disseminated intravascular coagulation (DIC), with up to 84% of women with acute renal failure also having DIC [8,10].

A study on the natural history of HELLP syndrome indicated that the worst biochemical state was reached within 24 to 48 h after delivery [11]. Our case reinforces the important clinical lesson that even after this period a high level of suspicion needs to occur with all women, regardless of their blood pressure, in order to enable timely intervention for a serious clinical condition associated with significant maternal morbidity.

4. Conclusion

HELLP syndrome is associated with significant morbidity and mortality. A high clinical suspicion is required for this condition, even in the late postpartum period.

Conflict of interest

No author has a conflict of interest.

Author's contributions

Natasha Louise Pritchard.

Group1 – conception and design, acquisition of data, analysis and interpretation of data.

Group 2 – drafting the article and critical revision of the article.

Group 3 – final approval of the version to be published.

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Group1 – analysis and interpretation of data.

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References

- [1] Haram K, Svendsen E, Abildgaard U. The HELLP syndrome: clinical issues and management. A Review. *BMC Pregnancy Childbirth* 2009;9(8). <http://dx.doi.org/10.1186/1471-2393-1189-1188>.
- [2] Clarke S, Nelson-Piercy C. Pre-eclampsia and HELLP syndrome. *Anaesth Intensive Care Med* 2008;9(3):110–4.
- [3] Sibai B, Ramadan M, Usta I, Salama M, Mercer B, Friedman S. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). *Am J Obstet Gynecol* 1993;169(4):1000–6.
- [4] Matthys L, Coppage K, Lambers D, Barton J, Sibai B. Delayed postpartum preeclampsia: an experience of 151 cases. *Am J Obstet Gynecol* 2004;190:1464–6.
- [5] Siba B. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. *Obstet Gynecol* 2004;103(5Pt1):981–9.
- [6] Sibai B. The HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): much ado about nothing? *Am J Obstet Gynecol* 1990;162(2):311–6.
- [7] Martin J, Rinehart B, May W, Magann E, Terrone D, Blake P. The spectrum of severe preeclampsia: comparative analysis by HELLP (hemolysis, elevated liver enzyme levels, and low platelet count) syndrome classification. *Am J Obstet Gynecol* 1999;180(6):1373–84.
- [8] Haddad B, Barton J, Livingston J, Chahine R, Sibai B. Risk factors for adverse maternal outcomes among women with HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome. *Am J Obstet Gynecol* 2000;183(2):444–8.
- [9] Pop-Tranjkovic, Antic V, Kopitovic V, Popovic J, Trenkic M, Vacic N. Postpartum HELLP syndrome – the case of lost battle. *Ups J Med Sci* 2013;118:51–3.
- [10] Sibai B, Ramadan M. Acute renal failure in pregnancies complicated by hemolysis, elevated liver enzymes, and low platelets. *Am J Obstet Gynecol* 1993;168:1682–90.
- [11] Martin J, Blake P, Kenneth P, James M, Hess L, Martin R. The natural history of HELLP syndrome: patterns of disease progression and regression. *Am J Obstet Gynecol* 1991;164(6):1500–13.

Suggested Reading

- [12] Weinstein L. It has been a great ride: the history of HELLP syndrome. *Am J Obstet Gynecol* 2005;193(3):860–3.
- [13] Cakmak B, et al. Late postpartum HELLP syndrome 60 hours after delivery associated with mild preeclampsia. *J Clin Diagn Res* 2013;7(12):2998–9.
- [14] Stella C, Sibai B. Preeclampsia: diagnosis and management of the atypical presentation. *The J Matern Fetal Neonatal Med* 2006;19(7):381–6.
- [15] Poourrat O, et al. Differentiation between severe HELLP syndrome and thrombotic microangiopathy, thrombotic thrombocytopenic purpura and other imitators. *Eur J Obstet Gynecol Reprod Biol* 2015;189:68–72.
- [16] Audibert F, et al. Clinical utility of strict diagnostic criteria for the HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome. *Am J Obstet Gynecol* 1996;175(2):460–4.